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Advances in antithrombotic therapy of acute myocardial infarction.

The Dawn Of A New Era In The Treatment Of Acute Myocardial Infarction

American Heart Journal. 138(2, Part 2) Supplement:S171-S176, August 1999.
Gensini, G. F. MD; Comeglio, M. MD; Falai, M. MD

Abstract:
The aim of the acute treatment of myocardial infarction is to restore, as promptly as possibl vessel. Thrombolysis is a cornerstone of treatment, and direct coronary angioplasty (PTCA) even better alternative reperfusion strategy. The activation of hemostasis after plaque disru represents a strong rationale for the use of antithrombotic drugs. The results of the ISIS-2 t Antiplatelet Trialists' Collaboration indicated that aspirin is mandatory in patients with acute secondary prevention. Recently, the efficacy of abciximab and other glycoprotein IIb/IIIa inh treatment of acute coronary syndromes and after PTCA, and their early use in patients with is presently under evaluation. Anticoagulation with heparin appears to be only slightly effect infarction not treated with thrombolysis; however, a rationale exists for its use in patients un and/or surgical revascularization and in conjunction with fibrin-specific thrombolytic agents. way on the possible usefulness of low-molecular-weight heparin. Direct antithrombin agents have been recently studied as an adjunct to thrombolysis. The data from these studies indic therapeutic window, with only marginal advantage over heparin; studies with newer compou still a mandatory drug in patients with acute myocardial infarction; the most promising agent glycoprotein IIb/IIIa inhibitors. Heparin and low-molecular-weight heparins are indicated in s studies are needed to assess the value of newer direct thrombin inhibitors.

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1: Herz. 1996 Feb;21(1):44-59. Related Articles, Links

-Differential antithrombotic therapy in patients with low and high PTCA risk-.

[Article in German]

Silber S, **Dorr R**.

Herzkatheterlabor der Kardiologischen Gemeinschaftspraxis, Klinik Dr. Muller, Munchen.

Acute coronary occlusion as well as restenosis still represent the major limitations of coronary interventions. Either event seems to be related to thrombus formation. The purpose of this overview is to summarize the current status of the usefulness of conventional and newer antithrombotic drugs regarding the prevention of acute occlusion and restenosis (excluding stents). ANTICOAGULATION: For ethical reasons, no placebo-controlled studies were conducted to prove the usefulness of heparin in preventing acute occlusions. The dosage mostly used is 10,000 U, although a relationship between dosage and complication rate has not been documented. A prolonged heparin infusion in patients with low risk and uncomplicated PTCA has no advantages. Restenosis is not influenced by prolonged infusion of heparin or administration of coumadin as well. Low molecular weight heparin is currently under investigation. Hirudin and hirulog have shown promising results with less acute occlusions; however, their therapeutic range must be considered. ANTIAGGREGATION: In controlled studies, ASA significantly reduced acute occlusions during PTCA when given in addition to heparin. Ticlopidin is as effective as ASA, but due to its side effects should only be administered when contraindications to ASA exist. ASA significantly reduced restenosis in only 1 of 4 studies with limited number of patients. Thromboxane inhibitors such as ridogrel or clopidogrel showed promising initial results. Trapidil significantly reduced restenosis in 2 studies; quantitative stenosis analysis, however, was not performed. Inhibition of platelets by glycoprotein (GP) IIb/IIIa receptor antagonists represents an innovative therapeutic concept: numerous controlled trials have documented a significant reduction in cardiac ischemic events and therefore indirectly in restenosis rates. The recombinant monoclonal antibody c7E3 Fab seems to be more effective than the synthetic integrelin. Unfortunately, efficacy appears to be in direct relationship to the risk of bleeding complications. The clinical role of oral GP IIb/IIIa inhibitors has yet to be established. For patients with high risk PTCA, the use of hirudin instead of heparin as well as the addition of GP IIb/IIIa inhibitors should be considered.

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